

to those with the AA genotype.<sup>9</sup> The groups were not, however, matched by socioeconomic and other factors that might have contributed to the observed difference, and the selection of subjects from twin studies may have further complicated assessment. A study from Jamaica (p 1371) appears to reach different conclusions. Both investigations concerned small numbers of people, and clearly more extensive studies are required; but the prevalence of the trait and the far-reaching implications of any possible pathogenicity demand that these should be based on impeccable diagnostic and epidemiological criteria.

<sup>1</sup> Smith, E W, and Conley, C L, *Bulletin of Johns Hopkins Hospital*, 1955, **96**, 35.

<sup>2</sup> Rywlin, A M, and Benson, J, *American Journal of Clinical Pathology*, 1961, **36**, 142.

<sup>3</sup> O'Brien, R T, *et al*, *New England Journal of Medicine*, 1972, **287**, 720.

<sup>4</sup> Whalley, P J, Martin, F G, and Pritchard, J A, *Journal of the American Medical Association*, 1964, **189**, 903.

<sup>5</sup> Ashcroft, M T, Miall, W E, and Milner, P F, *American Journal of Epidemiology*, 1969, **90**, 236.

<sup>6</sup> Allen, T D, *Journal of Urology*, 1964, **91**, 177.

<sup>7</sup> Atkinson, D W, *Blood*, 1969, **34**, 736.

<sup>8</sup> Serjeant, G, and Gueri, M, *British Medical Journal*, 1970, **1**, 820.

<sup>9</sup> McCormack, M K, *et al*, *Paediatrics*, 1975, **56**, 1021.

## Radiotherapy and the heart in Hodgkin's disease

Many treatments in medicine and surgery are not totally safe. To put a risk into perspective we need to study its incidence in various circumstances and, in the case of drugs or radiation, at various dosage levels. Every risk and every hope of benefit must be weighed against the hazards and benefits of alternative courses of action (or inaction). If we are too complacent, patients may be put at risk before the danger is fully appreciated. If we are too alarmist, those who would benefit from a treatment may be denied it because of exaggerated fears of its effects.

A recent report in the *American Journal of Medicine*<sup>1</sup> has described two fairly young men who had coronary attacks some years after successful treatment for Hodgkin's disease, and one of them died. Was mediastinal radiotherapy responsible? While agreeing that other factors may have played a part (one of them had smoked 40 cigarettes a day), the American authors argued that it probably was. They had found four other reports of young men who had died in similar circumstances, necropsy confirming that coronary disease, not recurrent Hodgkin's disease, had caused death.

Isolated reports of this kind serve a useful purpose but may easily give a false impression of the size of the problem. Reviews<sup>2-4</sup> of all the experimental and clinical evidence, undertaken over the last 50 years, have concluded that the heart stands up surprisingly well to radiation, often showing not the slightest electrocardiographic or histological abnormality even after absorbing large doses. When electrocardiographic changes have occurred they have been mostly trivial and transient and sometimes due to other causes.<sup>3</sup> Many thousands of patients with breast cancer have survived in good health for many years after quite intensive radiation to part of the heart in the course of radiotherapy to the parasternal lymph glands. No evidence has been found of any increased incidence of heart disease in these women.

When the mediastinal glands are irradiated in Hodgkin's disease the dose absorbed by the heart (or a portion of it) will

vary considerably, not only with the dose chosen for the target lymph glands but also according to the technique. For this reason the incidence of cardiac effects reported from one centre (for example, 6% at Stanford University<sup>4</sup>) may not apply to another. Many centres seem to avoid these complications completely, perhaps partly by giving a dose which others might regard as less than optimal. At the other extreme, Byhardt *et al*<sup>5</sup> recently reported no fewer than 24 cases of pericardial effusion in 83 patients given mediastinal radiation. Nevertheless, 10 out of the 24 were symptomless; Hodgkin's disease itself occasionally affects the pericardium; and radiation was given mainly by a single anterior beam giving an average pericardial dose of 5325 rads. This was more than the mediastinal glands themselves received and is considerably above the dose received by the heart when treated by some of the techniques commonly used in other centres.

Patients with existing heart disease might be thought to be especially at risk, but there is no evidence that this is so. Indeed, controlled experiments in dogs have actually shown a beneficial effect, radiated animals showing a higher survival rate after coronary artery ligation.<sup>6</sup>

Hence probably there is no good evidence that heart muscle and its blood supply are any more susceptible to radiation than any other muscle. Most radiotherapy centres use a dose technique for mediastinal Hodgkin's disease which probably carries only a small risk of contributing to future heart disease. But vigilance and further study are required, preferably always with expert assessment of the dose received at different points in the heart and mediastinum.

<sup>1</sup> McReynolds, R A, Gold, G L, and Roberts, W C, *American Journal of Medicine*, 1976, **60**, 39.

<sup>2</sup> Jones, A, and Wedgwood, J, *British Journal of Radiology*, 1960, **33**, 138.

<sup>3</sup> Biran, S, Hochmann, A, and Stern, S, *Clinical Radiology*, 1969, **20**, 433.

<sup>4</sup> Stewart, J R, and Fajardo, L F, *Radiologic Clinics of North America*, 1971, **9**, 511.

<sup>5</sup> Byhardt, *et al*, *Cancer*, 1975, **35**, 795.

<sup>6</sup> Senderoff, E, *et al*, *Proceedings of the Society for Experimental Biology and Medicine*, 1959, **100**, 1.

## Boys who are too tall

Being too short or too tall may be a social or psychological disability—and sometimes both. Yet it is easier to find a consensus of opinion on a height that is too short for psychological comfort than to determine what would be considered excessive. Last year we reviewed<sup>1</sup> attempts to limit the height of healthy girls whose growth promised to make them excessively tall. More recently Zachmann *et al*<sup>2</sup> have attempted to limit the growth of boys whose predicted height was over 198 cm.

Clearly the success of attempts to limit children's growth must depend on the accuracy with which eventual height can be predicted. The work of Tanner and his associates<sup>3</sup> does allow a usefully accurate prediction for normal children. Pathological causes of excess height, such as an over-secretion of growth hormone or cerebral gigantism, have to be excluded. For boys from 4 to 12 years the prediction of eventual height is accurate within  $\pm 7$  cm. Using this method, Zachmann's group found that the best results from treatment with testosterone came in boys at the onset of puberty whose bone age was about 12 years; they calculated that eventual height was curtailed by 8 cm. As they point out, their treatment was completely